- Group quarters at the community level that include the location, size, and type of nursing home facilities and mental health facilities.
- Major roads, state, metropolitan area, and county boundaries.
- Bus routes in select communities.

This model advances the state of disease transition modeling science by developing a set of documented tools that constitute an analog to a higher level language that supports the rapid development of large ABMs for studying problems analogous to disease transmission problems. This resource uses the above data resources defines above and it permits the properties of ABMs, the natural history of infectious diseases and the characteristics of the social networks that those diseases utilize transmit within to be studied experimentally.

*MIDAS – Models of Infectious Disease Agents Study.

PL-06 Examining the fourth stage of epidemiological transition in China: Some implications for health and health care provision

M.M. Islam*, L.J. Pei, X.M. Song, X.Y. Zheng. *Institute of Population Research, Peking University, Beijing-100871, China*

Today's China is experiencing the new epidemiological phase, characterized by increasing life expectancy and disease of affluence coupled with emerging and re-emerging diseases. Under this situation it is important to think strategically in the planning of health care provision of all population which is particularly important for rapidly modernizing present China. The aim of this paper is to analyze the fourth stage of epidemiological transition in current China based on existing theoretical framework by using descriptive statistical methods of analysis and to seek for some policy implications for health and health care provisions. Mortality data suggests that China has well entered to the fourth stage of epidemiological transition-the age of delayed degenerative diseases where Cardiovascular, cancer, respiratory and digestive diseases are the leading causes of death while the life expectancy has reached to more than 70 years. Study indicates that the increase in non-communicable diseases significantly increases the cost of illness and the burden of health care system. Moreover HIV/AIDS, and other emerging diseases like bird flu, SARS have become new threats to public health. Demographic and life style changes are likely to result in a substantial increase in non-communicable diseases in next coming years where it is an urgent priority to execute the policies to prevent the growing epidemics. Thus this paper examines the predominant major degenerative causes of death and the risk of dying from these diseases redistributing to older ages of people in China and to point out the new targets of health care policies under changing dilemmas.

PL-07 HIV and TGF-β1 regulates HCV replication through multiple pathways

W.Y. Lin*, E.M. Weinberg, R.X. Shao, L.F. Peng, R.T. Chung. *Gastrointestinal Unit, Massachusetts General Hospital, Harvard Medical School, Boston, MA 02114, USA*

Background: HIV co-infection increases HCV-related hepatic fibrosis progression, HCV persistence, and decreases response rates to interferon-based anti-HCV therapy. We have demonstrated that HIV increases HCV replication through enhancement of TGF-β1 expression. We sought to further explore the mechanism by which TGF-β1 regulates HCV through TGF-β1 signal pathways.
Methods: Huh7.5.1 cells were incubated with active or heat inactivated JFH1. Human TGF-β1 was tested for its effect on JFH1 uptake and replication. HCV levels in cell lysates were analyzed by HCV core ELISA and Q-PCR. TGF-β1 signaling was measured using a highly TGF-β1 responsive PAI1-Luc reporter construct. The effects of the ERK1/2 inhibitor U0126 and the PI3K inhibitor Ly294002 on JFH1 replication were analyzed.
Results: TGF-β1 increased active uptake of both active and inactivated JFH1 by over 3 fold at 4 hrs, and increased active JFH1 uptake and replication by over 5-fold at 24 hrs compared to untreated cells. U0126 decreased HCV replication by 67% and blocked TGF-β1 mediated PAI1 promoter signaling by 2.2-fold in Huh7.5.1 cells and 3.6-fold in JFH1-infected Huh7.5.1 cells. U0126 reduced TGF-β1 stimulated phospho-ERK1/2 in both uninfected and JFH1-infected Huh7.5.1 cells by Western blot. In contrast, Ly294002 did not block TGF-β1 mediated PAI1 promoter transcription or HCV replication enhancement.
Conclusions: Our data confirm that HIV appears to promote HCV replication through upregulating TGF-β1 in HCV-infected hepatocytes. TGF-β1 has a permissive effect on HCV uptake and replication. HCV replication enhancement takes place through both indirect (increasing viral uptake) and direct (regulation of HCV replication) means.

PL-08 Activation of VEGF-A/AKT/mTOR signal in ground glass hepatocytes and transgenic livers of Pre-S mutant: Implication for hepatitis B virus-related hepatocarcinogenesis

J.C. Yang[2]*, C.F. Teng[2], H.C. Wu[1], I.J. Su[1,2]. [1]*Division of Clinical Research, National Health Research Institutes, Tainan, Taiwan,* [2]*Graduate Institutes of Basic Medicine Sciences, National Cheng Kung University College of Medicine, Tainan, Taiwan*

Background and Aim: Ground glass hepatocytes (GGH) harbor HBV pre-S mutants in endoplasmic reticulum (ER) and represent the precursor lesions of hepatocellular carcinoma (HCC). Whether growth factors play a role in the progression from GGHs to HCC is investigated.
Methods: Growth factor(s) upregulated by pre-S mutants was identified using a cytokine/growth factor array in Huh-7 cells. Immunohistochemistry, RT-PCR, and Western blot analysis were performed to study the participation of these genes and their pathways in cells, liver tissues, and transgenic livers.
Results: Vascular endothelial growth factor (VEGF)-A was identified as the potential growth factor upregulated by pre-S mutants. By immunohistochemistry, GGHs and the transgenic mouse livers of pre-S2 mutant showed enhanced expression of VEGF-A. The VEGF-A upregulation by pre-S mutants could be suppressed by vomitoxin, an ER stress inhibitor. Furthermore, pre-S mutants-expressed Huh-7 cells exhibited activation of Akt/mTOR signaling and increased growth advantage, which could be inhibited by VEGF-A neutralization. Consistent with this notion, enhanced expression of VEGF-A and activation of Akt/mTOR signaling, comparable to the levels of paired HCC tissues, were also detected in HBV-related non-tumorous livers.
Conclusion: The enhanced expression of VEGF-A in GGHs, probably associated with pre-S mutants-induced ER stress, may provide a potential mechanism for the progression from GGHs to HCC through Akt/mTOR signaling in chronic HBV infection.